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Effects of Acute Respiratory Viral Infection on the Fetoplacental System of Pregnant Women

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Abstract: Fetoplacental insufficiency (FPI) is a complex of symptoms caused by various disorders of both the placenta and the fetus due to various diseases and obstetric complications. The variety of manifestations of FPN, the frequency and severity of complications for the pregnant woman and the fetus, the predominant violation of one or another function of the placenta depend on the duration of pregnancy, the strength, duration and nature of the impact of the injury. factors, as well as the degree of expression of the compensatory and adaptive abilities of the "mother-placenta-fetus" system at the stage of development of the fetus and placenta.

Keywords: FPN causes, pathogenesis, origin, diagnosis, prevention.

FPN can develop due to various reasons. Violations of placenta formation and functioning can be caused by diseases of the heart and vascular system of a pregnant woman (heart defects, circulatory failure, arterial hypertension and hypotension), kidneys, liver, lungs, blood pathology, chronic infection. , diseases of the neuroendocrine system (diabetes, hypo- and hyperfunction of the thyroid gland, pathology of the hypothalamus and adrenal glands) and a number of other pathological conditions. FPN in anemia is caused by a decrease in the level of iron both in the mother's blood and in the placenta itself, which leads to the inhibition of the activity of respiratory enzymes and the transport of iron to the fetus. In diabetes, the metabolism is disturbed, hormonal disorders and changes in the immune state are detected. Sclerotic damage of blood vessels leads to a decrease in arterial blood supply to the placenta. An important role in the development of FPN is played by various infectious diseases, especially those that occurred in an acute form or worsened during pregnancy. The placenta can be infected by bacteria, viruses, protozoa and other infectious agents.

In the formation of FPN, the pathology of the uterus is not important: endometriosis, myometrial hypoplasia, malformation of the uterus (saddle-shaped, two-horned). Uterine fibroids should be considered as a risk factor for FPN. However, the risk of FPN varies among pregnant women with uterine fibroids. The high-risk group includes primiparas aged 35 years and older, who are mainly located in the intermuscular location of large myomatous nodes, especially when the placenta is localized in the area where the tumor is located. The group with a low risk of FPN includes young women under 30 years old without severe extragenital diseases, who have small myomatous nodes in the fundus and body of the uterus, mainly subperitoneal.

Among the complications of pregnancy with FPN, gestosis takes the leading place. The risk of abortion should be considered both as a cause and as a consequence of FPN. When there is a risk of abortion, due to the different etiology of FPN, the pathogenesis of this complication has different options, and the prognosis for the fetus depends on the level of development of protective-adaptive reactions. With a low location or placenta previa, the vascularization of the subplacental zone is reduced. The thin wall of the lower segment of the uterus does not provide the necessary conditions for adequate vascularization of the placental bed and its normal functioning.

Relatively often, with this pathology, separation of the placenta occurs along with blood loss. Multiple gestation represents a natural pattern of FPN resulting from inadequate provision of the needs of two or more fetuses. The basis of FPN in the isoserological incompatibility of maternal and fetal blood often lies in the processes of maturation of the placenta. Anemia and hypoxia develop in the fetus, developmental delay occurs due to the disruption of protein synthesis processes and a decrease in enzyme activity. The functional state of the placenta is largely determined by its level of development in accordance with the period of pregnancy and the maintenance of protective and adaptive mechanisms. Adapting the maturity of the placenta to the period of pregnancy is one of the most important conditions for the adequate development of the fetus and its protection. There is no doubt that the late age of a pregnant woman (over 35 years old) or, on the contrary, young age (under 17 years old), a history of serious illness (abortions, inflammatory diseases), bad habits, the influence of adverse environmental factors. , poor nutrition, social vulnerability and household disorder also contribute to the complex formation of the placenta and its dysfunction [1].

Clinical practice and scientific research results show the multifactorial nature of FPN. In this regard, it is almost impossible to identify any factor in the development of this complication. The listed pathological conditions do not equally affect the development of FPN. Often, several etiological factors are involved in the development of this pathology, one of which can be the leader. Pathological changes that occur during FPN lead to a decrease in uteroplacental and fetoplacental blood flow; decreased arterial blood supply to the placenta and fetus; restriction of gas exchange and metabolism in the fetoplacental complex; disruption of placental ripening processes; decreased synthesis and balance of placental hormones. All these changes suppress the compensatory-adaptive capabilities of the "mother-placenta-fetus" system, slow down the growth and development of the fetus, lead to a complicated course of pregnancy and childbirth (premature termination of pregnancy, gestosis, early and premature birth threat). late birth, birth anomalies, early placental abruption, etc.) [5].

Fetal hypoxia naturally develops as a result of exposure to harmful factors and implementation of pathogenetic mechanisms leading to FPN. In the initial stages, vasopressor factors are activated in the fetus, the tone of peripheral vessels increases, tachycardia is noted, the frequency of respiratory movements increases, motor activity increases, and cardiac output increases.

Further development of hypoxia causes tachycardia to be replaced by bradycardia, arrhythmia appears and cardiac output decreases. The adaptive response to hypoxia is a redistribution of blood in favor of the brain, heart, and adrenal glands, while reducing blood supply to other organs. At the same time, motor and respiratory activity of the fetus is inhibited [5].

Classification of FPN

Taking into account the state of protective-adaptive reactions, it is recommended to classify FPN as compensated, subcompensated, decompensated [3].

The compensated form of FPN is characterized by the initial manifestations of the pathological process in the fetoplacental complex. Protective and adaptive mechanisms are activated and undergo a certain tension, which creates conditions for the further development of the fetus and the development of pregnancy. With adequate therapy and labor management, a healthy baby can be born.

The subcompensated form of FPN is characterized by an increase in the severity of the complication. Protective-adaptive mechanisms are under extreme stress (fetoplacental complex capabilities are almost exhausted), which does not allow them to perform adequately for an adequate course of pregnancy and fetal development. The risk of complications for the fetus and newborn increases.

In the decompensated form of FPN, there is excess tension and a violation of compensatory-adaptive mechanisms, which no longer provide the necessary conditions for the further normal development of pregnancy. Irreversible morphofunctional disorders appear in the fetoplacental system. The risk of developing serious complications for the fetus and newborn (including their death) increases significantly. The clinical manifestation of FPN manifests itself in the violation of the basic functions of the placenta.

Intrauterine growth restriction

Changes in the respiratory function of the placenta are mainly indicated by signs of fetal hypoxia. In this case, the pregnant woman first pays attention to the increased (incorrect) motor activity of the fetus, and then to its decrease or complete absence. The most characteristic symptom of chronic FPN is intrauterine growth retardation. The clinical manifestation of intrauterine growth retardation is a decrease in the volume of the pregnant woman's abdominal cavity (abdominal circumference, height of the uterine fundus) compared to the normal parameters typical for a certain period of pregnancy.

With a symmetrical form of intrauterine growth that develops from the early stages of pregnancy, there is a proportional delay in the length and weight of the fetus. At the same time, the quantitative indicators of fetometry have lower values compared to the individual fluctuations characteristic of a certain period of pregnancy.

Asymmetric form of intrauterine growth retardation is characterized by disproportionate development of the fetus. This form often occurs in the second or third trimester of pregnancy and is manifested by a delay in fetal body weight with its normal length. The size of the abdomen and chest of the fetus is mainly reduced, which is associated with a delay in the development of parenchymal organs (primarily the liver) and subcutaneous fat tissue. The size of the head and limbs of the fetus corresponds to the indicators typical for this stage of pregnancy.

Placental dysfunction

A reflection of the violation of the protective function of the placenta when the placental barrier is weakened is intrauterine infection of the fetus under the influence of pathogenic microorganisms that enter the placenta. It is also possible that various toxic substances penetrate the placental barrier, which also has a harmful effect on the fetus.

A change in the synthetic function of the placenta is accompanied by an imbalance in the level of hormones it produces and a decrease in protein synthesis, which is manifested by (long) intrauterine growth retardation, hypoxia, and the pathology of uterine contractile activity during pregnancy and childbirth.. -premature termination of pregnancy, premature birth, birth abnormalities).

A long and frequent increase in myometrial tone leads to a decrease in arterial blood flow to the placenta and causes venous stagnation. Hemodynamic disorders reduce the gas exchange between the mother's body and the fetus, which prevents the supply of oxygen and nutrients to the fetus, the removal of metabolic products, and contributes to the increase of fetal hypoxia.

Violation of the endocrine function of the placenta can also lead to post-term pregnancy. A decrease in the hormonal activity of the placenta causes the dysfunction of the vaginal epithelium, which creates favorable conditions for the development, exacerbation of infection or the appearance of inflammatory diseases of the urogenital tract. Against the background of impaired excretory function of the placenta and amniotic membranes, a pathological amount of

amniotic fluid is observed - often in oligohydramnios and in some pathological conditions (diabetes mellitus, hemolytic disease of the fetus, intrauterine infection). etc.) - polyhydramnios.

Diagnosis of FPN

In the initial stage of development of FPN, the listed clinical signs may be weakly expressed or absent. In this regard, methods of laboratory and instrumental dynamic monitoring of the condition of the fetoplacental complex in the high-risk group for the development of FPN are important. The dominant position in the clinical picture can be taken by symptoms of the main disease or an advanced complication of FPN. The severity of FPN and the impairment of compensatory and adaptive mechanisms directly depend on the severity of the underlying disease and its duration. FPN takes its most severe course when pathological symptoms appear before or after 30 weeks of pregnancy. Thus, the most complete information about the form, nature, severity of FPN and the severity of compensatory-adaptive reactions can be obtained from complex dynamic diagnostics [2].

Considering the multifactorial etiology and pathogenesis of FPN, its diagnosis should be based on a comprehensive examination of the patient. To make a diagnosis of FPN and to determine the causes of this complication, it is necessary to pay great attention to the correct collection of anamnesis. During the interview, the patient's age (late or young primary woman), her health characteristics, previous extragenital, neuroendocrine and gynecological diseases, surgical interventions, bad habits are assessed, her profession, conditions and lifestyle are clarified. .

Information about the characteristics of menstrual function, the period of its formation, the number and course of previous pregnancies is of great importance. Menstrual dysfunction is a reflection of the pathology of neuroendocrine regulation of reproductive function. It is important to assess the progress of the current pregnancy, the nature of obstetric complications and, most importantly, the presence of diseases that develop during pregnancy (arterial hypertension or hypotension, kidney, liver pathologies, diabetes, anemia, etc.).). You should pay attention to the complaints of a pregnant woman: increased or suppressed motor activity of the fetus, pain in the lower abdomen, increased tone of the uterus, atypical discharge from the genital tract, which may be accompanied by an unpleasant odor. odor and itching.

During an objective examination, the condition of the uterine tone is assessed by palpation. Uterine fundal height and abdominal circumference are measured and compared to the pregnant woman's body weight and height, as well as the estimated gestational age. Such measurements are important and at the same time the simplest indicators for diagnosing intrauterine growth retardation, oligohydramnios and polyhydramnios. During the external examination of the genitals and examination using a mirror, it is necessary to pay attention to the presence of signs of inflammation, the nature of discharge from the genitals, and to take material from the vaginal wall and cervical canal. taken from the urinary tract for microbiological and cytological examination [2].

During the ultrasound examination, the size of the fetus (the size of the head, torso and limbs) is determined and compared with the standard indicators typical of the expected gestational age. To determine whether the size of the fetus corresponds to the expected gestational age and to determine intrauterine growth retardation, the basis of ultrasound diagnostics is the comparison of fetometric indicators with normative data. An indispensable condition is the evaluation of the anatomical structures of the fetus to identify abnormalities in its development. Ultrasound examination also includes the placenta. In this case, the localization of the placenta, the thickness of the placenta, the distance of the placenta from the internal os, the degree of maturation of the placenta in accordance with the period of pregnancy, pathological inclusions in the structure of the placenta, the location of the placenta in relation to myomatous nodes or the scar in the uterus are determined. During the study, the volume of amniotic fluid, the structure of the umbilical cord and its location in the umbilical cord are evaluated [4].

Doppler ultrasound examination is a very informative, relatively simple and safe diagnostic method, which can be used for comprehensive dynamic monitoring of the state of blood circulation in the "mother-placenta-fetus" system after 18-19 weeks of pregnancy, because by this time the second phase of cytotrophoblast invasion wave has ended. The nature of hemodynamics in the umbilical arteries makes it possible to assess the state of fetoplacental blood flow and microcirculation in the fetal part of the placenta. In order to determine the violation of uteroplacental blood flow, studies are carried out in the uterine arteries of both sides [4].

Cardiotocography (CTG) is an important component of the comprehensive assessment of the state of the fetus, which is a method of functional assessment of the state of the fetus based on the frequency of heartbeats and uterine contractions, the influence of external stimuli or their changes. activity of the fetus itself. CTG significantly expands the possibilities of antenatal diagnosis, which allows to solve the issues of rational pregnancy management tactics.

Treatment of pregnant women with FPN

If FPN is detected, it is recommended to hospitalize the pregnant woman for thorough examination and treatment. Pregnant women with a compensated form of FPN can be excluded if the started treatment has a positive effect and there are necessary conditions for dynamic clinical and instrumental monitoring of the nature of pregnancy and the effectiveness of therapy. The leading place in the implementation of therapeutic measures is the treatment of the main disease or the complication of FPN. Currently, unfortunately, it is not possible to get rid of FPN completely with the help of any therapeutic interventions. The used therapy tools only help to stabilize the existing pathological process and preserve the compensatory and adaptive mechanisms to the extent that allows the pregnancy to continue until the optimal possible date of birth.

The basis of the treatment of placental insufficiency is measures aimed at improving uteroplacental and fetoplacental blood flow. Medicines used for this expand blood vessels, relax uterine muscles and improve the rheological properties of blood in the "mother-placenta-fetus" system [2].

Treatment of FPN should be aimed at improving uteroplacental and fetoplacental blood flow; increased gas exchange; correction of rheological and coagulation properties of blood; elimination of hypovolemia and hypoproteinemia; normalization of vascular tone and contractile activity of the uterus; strengthen antioxidant protection; optimization of metabolic and metabolic processes. A standard treatment scheme for FPN cannot exist due to the individual combination of etiological factors and pathogenetic mechanisms for the development of this complication. The choice of drugs should be made individually for each specific observation and should be differentiated taking into account the severity and duration of the complication, etiological factors and pathogenetic mechanisms based on this pathology. Doses of drugs and the duration of their use require an individual approach. Attention should be paid to correcting the side effects of some drugs.

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